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Original Article

Physiotherapy Section

Effect of Electrical Muscle Stimulation and Resistance Training on the Lipid Profile in Sedentary Type-II Diabetic Individuals: An Experimental Study

VISHWAJEET TRIVEDI[1], NEELIMA MISHRA[2], KAMRAN ALI[3]

ABSTRACT

Introduction: Diabetes Mellitus (DM) is associated with dyslipidaemia, a major contributor to cardiovascular complications. Resistance Training (RT) is known to improve lipid metabolism, but adherence can be challenging. Electrical Muscle Stimulation (EMS) has emerged as a potential alternative, promoting muscle activation and metabolic improvements.

Aim: To evaluate and compare the effects of EMS and RT on lipid profile parameters, including Total Cholesterol (TC), Low Density Liopprotein Cholesterol (LDL-C), High Density Liopprotein Cholesterol (HDL-C) and Triglycerides (TG), in sedentary individuals with diabetes.

Materials and Methods: An experimental study with a pretest/post-test design was conducted in the Department of Physiotherapy at GD Goenka University, Gurugram, Haryana, India starting from October 2023 until September 2024. A total of 66 sedentary type II diabetic subjects (both males and females) with a fasting blood glucose level between 100 mg/dL and 250 mg/dL and on oral hypoglycaemic drugs, without any major systemic or diabetic complications, were included in the study. Subjects were assigned to three groups through a convenient sampling method: EMS, RT and a control group. The EMS group, with a mean age of 53.0±3.7 years, received Russian current stimulation on three alternate days per week for 12 weeks. The RT group, with a mean age of 52.0±4.84 years, performed progressive resistance exercises on three alternate days per week for 12 weeks, while the control group, with a mean age of 49.77±6.75 years, received standard patient education on diet and physical activity. Lipid profile parameters, including TC, HDL-C, LDL-C, Very Low Density Liopprotein Cholesterol (VLDL-C), TGs and cholesterol/HDL ratio, were assessed at baseline, postintervention (12 weeks) and after a three-month follow-up. The pre-post data for intervention groups were analysed using Analysis of Variance (ANOVA) at three time points with p-value <0.05.

Results: Repeated measures ANOVA showed significant group-time interactions for TC (p-value <0.001, η^2=0.348), LDL-C (p-value=0.003, η^2=0.120), triglycerides (p-value <0.001, η^2=0.213), HDL-C (p-value <0.001, η^2=0.162) and cholesterol/HDL ratio (p-value=0.002, η^2=0.194), while changes in VLDL were not significant (p-value=0.713). Pairwise comparisons indicated significantly lower TC, LDL-C and cholesterol/HDL ratio in the EMS and RT groups compared to controls (p-value <0.01), with no difference between EMS and RT (p-value >0.05). Triglycerides decreased significantly in both intervention groups (p-value <0.05), with EMS showing a greater reduction, though not statistically different from RT (p-value=1.000). HDL-C changes were minor and nonsignificant across groups (p-value>0.05). These findings suggest that both EMS and RT effectively improve lipid profiles, with RT favouring cholesterol modulation and EMS being more effective for triglyceride reduction.

Conclusion: Both EMS and RT effectively improved lipid profiles in individuals with Type 2 Diabetes Mellitus (T2DM), with RT demonstrating superior benefits in reducing cholesterol and LDL-C, while EMS was more effective in lowering triglycerides. EMS may serve as a viable alternative for individuals with exercise limitations, offering a non pharmacological approach in managing diabetic dyslipidaemia.

Keywords: Cholesterol, Diabetes mellitus-type 2, Electric stimulation therapy, Lipid metabolism

INTRODUCTION

The DM is a chronic metabolic disorder characterised by persistent hyperglycaemia due to insulin resistance or deficiency. It is a major global health concern, contributing to cardiovascular diseases, neuropathy, nephropathy and retinopathy [1]. One of the critical complications of diabetes is dyslipidaemia, which involves an imbalance in lipid parameters, including elevated LDL-C,VLDL-C and TG, along with reduced HDL-C [2]. Insulin resistance, intensified by hyperglycaemia, is the main cause of dyslipidaemia in individuals with T2DM. Insulin resistance promotes the production of Triglyceride-Rich Lipoprotein (TRL) in the liver and intestines, as well as lipolysis in adipose tissue [3]. Dyslipidaemia is exacerbated by elevated inflammatory adipokines and causes an increase in free fatty acid flow as a result of insulin resistance [4]. Approximately 80% of all diabetic fatalities are caused by atherosclerosis, with 75% attributable to coronary atherosclerosis and 25% to peripheral or cerebral vascular disease [5]. Additionally, more than 75% of all hospitalisations for complications related to diabetes are caused by atherosclerosis and vice versa [6]. Diabetic dyslipidaemia significantly contributes to the increased risk of atherosclerosis, myocardial infarction and stroke in diabetic patients. In order to minimise the risk of cardiovascular disease in people with T2DM, the treatment of dyslipidaemia is prioritised [7].

Now-a-days, lipid-lowering medications are available that not only effectively reduce LDL-C but also do not increase the risk of New-Onset Diabetes (NOD) or glucose impairment. In fact, several of these medications may even help regulate blood sugar levels [8]. Despite these medications, lipid abnormalities are often observed in people with T2DM, even with good glycaemic control [9]. Furthermore, pharmacological innovations and lipid-lowering options

have progressed rapidly, the cost and accessibility of these drugs have fallen significantly behind [10]. Thus, continuity of care and adherence to guidelines-directed treatment modalities are essential for long-term patient wellbeing. Lifestyle interventions, including exercise and diet, are considered primary therapeutic strategies for improving lipid metabolism in diabetic individuals [11,12].

Traditional RT has been widely recognised for its role in enhancing insulin sensitivity, improving lipid profiles and reducing cardiovascular risks in patients with T2DM [7]. However, adherence to conventional exercise programmes can be challenging due to physical limitations, lack of motivation, or time constraints [8]. In recent years, EMS has emerged as a promising alternative or adjunct to conventional RT. EMS is a neuromuscular stimulation technique that induces involuntary muscle contractions via externally applied electrical impulses, mimicking voluntary muscle contractions and promoting muscular adaptations [9]. EMS has been found to improve muscle strength, enhance metabolic rate and influence lipid metabolism, making it a potential therapeutic approach for individuals with diabetes and limited exercise capacity [10].

The role of exercise in the management of diabetes-related lipid abnormalities has been well established, with RT demonstrating positive effects on lipid metabolism, glucose homeostasis and cardiovascular health [11]. RT enhances muscle mass, which plays a crucial role in glucose uptake and lipid oxidation, thereby reducing circulating triglycerides and LDL-C while increasing HDL-C levels [12]. Despite many benefits, numerous diabetic individuals face barriers in engaging in structured RT programmes due to co-morbidities, joint pain, or fatigue [13].

EMS has been proposed as an alternative to traditional exercise modalities, as it activates muscle fibres and induces metabolic changes similar to voluntary contractions, potentially benefiting lipid metabolism and glucose regulation [14]. Previous studies suggest that EMS can lead to significant improvements in body composition, insulin sensitivity and lipid profile markers in sedentary and diabetic populations [15-18]. Despite increasing interest in the effects of EMS and RT on metabolic health, there is a lack of comparative studies examining their efficacy in improving lipid profiles in sedentary individuals with T2DM. This study aimed to evaluate and compare the effects of EMS and RT on lipid profile parameters, including TC, LDL-C, HDL-C and TG, in individuals with T2DM.

The null hypothesis (H_0) states that there is no significant difference in the effects of EMS and RT on lipid profile parameters in individuals with T2DM. The alternative hypothesis (H_1) posits that EMS and RT result in distinct effects on lipid profile parameters in this population.

MATERIALS AND METHODS

An experimental study with a pretest/post-test design, including a follow-up three months after the termination of the intervention, was conducted at the Department of Physiotherapy in the School of Healthcare and Allied Sciences at GD Goenka University, Gurugram, Haryana, India from October 2023 to September 2024. The study was approved by the Institutional Ethical Committee (WWET/2023/IEC-AP/03) and registered with the Clinical Trials Registry of India (CTRI/2023/09/057825). This study investigated independent variables such as EMS and resistance exercise on dependent variables like lipid profiles in sedentary individuals with T2DM. The reference ranges for various lipid profile parameters are as follows: TC (<200 mg/dL), HDL (40-60 mg/dL), LDL (<100 mg/dL), VLDL (<40 mg/dL) and TG (<150 mg/dL) [19]. No modifications were made to the prescribed drug regimen for diabetes and hyperlipidaemia management in the study participants.

Inclusion criteria: Individuals aged 45 to 65 years diagnosed with T2DM, with fasting blood glucose levels ranging between 100 and 250 mg/dL. Both male and female participants were recruited, provided they had no major systemic or diabetic complications. Only those using oral hypoglycaemic agents, but not insulin, were included. Participants with a low activity level, as assessed by the International Physical Activity Questionnaire (IPAQ) criteria [20], were considered eligible. Additionally, subjects were required to be cooperative and willing to participate were included in the study.

Exclusion criteria: Participants with T1DM who were using insulin or any form of injection, pregnant women, individuals who were physically active and those with uncontrolled or fluctuating hypertension or blood glucose levels. A history of myocardial infarction, any condition that could increase the risk or interfere with the study or data evaluation, cardiac arrhythmias, unwillingness to cooperate, and the presence of metal implants, cardiac pacemakers, or other electrical devices contraindicated for electrical stimulation. Moreover, subjects who had undergone major surgical interventions, those with skin conditions that might hinder the application of electrical stimulation, and individuals with substance abuse issues (alcohol, smoking, or drugs affecting the neuromuscular system) were also excluded from the study.

Sample size: A total sample size of 66 potential subjects was screened and recruited through convenient sampling by an expert in Internal Medicine, based on the inclusion and exclusion criteria. All participants were divided equally into three groups: Experimental group (n=22), Exercise group (n=22) and Control group (n=22).

Sample size calculation was conducted using the following formula [21]:

$$n=\frac{\{z\alpha/2+z\beta\}^2\times 2\sigma^2}{\delta^2}$$

where:

- σ of HbA1c=0.7
- Significance level 5%
- Power=80%
- Hypothesis=two tail
- zα=1.96
- zβ=0.84 [22]

n=required sample size per group

$Z_{\alpha/2}$=critical value of the standard normal distribution corresponding to the desired significance level (α);

Z_{β}=critical value of the standard normal distribution corresponding to the desired power (1-β);

σ=standard deviation of the outcome variable;

δ=minimum detectable difference between the two group means.

Study Procedure

A multi-channel (8 channels) electrical muscle stimulator from Johri Digital (model no. TR841) was employed for the experimental group to administer stimulation to six muscles (bilateral glutei maximus, hamstrings and quadriceps) during a single 30-minute session, with three sessions per week over a 12-week period. Following a standard clinical protocol, a Russian current (medium frequency current) at a frequency of 2500 Hz and motor-level intensity was applied. The stimulation protocol included 10 seconds of stimulation followed by a 50-second rest interval, with two muscles being stimulated simultaneously for 10-minute increments per muscle pair.

A supervised Progressive Resistance Training (PRT) protocol was implemented for the exercise group, targeting the hip extensors, hip flexors, knee extensors, knee flexors, elbow flexors, shoulder flexors and plantar flexors bilaterally. Each participant began with a 10-minute warm-up, consisting of gentle stretching for all four limbs. This was followed by resistance exercises, comprising up to two sets of 10 repetitions per muscle group, using a resistance equivalent to three Repetition Maximum (RM) in each session. The training sessions were conducted on three alternate days per week

for a duration of 12 weeks. The 1RM (One RM) was calculated using the formula:

1RM=weight lifted/1.0278 - (repetitions×0.0278)

as outlined by Brzycki, 1993 [23]. When participants were able to perform 20 repetitions with ease, an additional 0.5 kg of resistance was added to the training load [24].

Participants in the control group received educational interventions on dietary management, exercise and glycaemic control strategies. They were encouraged to increase their leisure physical activity and participate in low-to-moderate intensity exercises, such as walking, household chores and cycling, for 20-30 minutes.

STATISTICAL ANALYSIS

The outcome measures, including TC, LDL, HDL, VLDL, TG and the Cholesterol/HDL ratio, were analysed at three time points using the Statistical Package for the Social Sciences (SPSS) software, version 25.0 (IBM, USA). Descriptive statistics were applied to calculate the mean and standard deviation for the demographic and anthropometric profiles, as well as the outcome measures of the participants. The Shapiro-Wilk test was employed to assess the normality of the data. Group differences were analysed using repeated measures ANOVA and the corresponding p-values and F-values were computed. Post-hoc tests were conducted to examine specific group comparisons, with statistical significance set at p-value <0.05.

RESULTS

The study analysed the effects of electrical stimulation, RT and control interventions across the groups. The pre-post data for the intervention groups was analysed using ANOVA at three time points. [Table/Fig-1] presents the baseline characteristics of the study participants across the three groups: Experimental, Exercise and Control, each comprising 22 individuals. The mean age of the experimental group was 53±3.7 years, while the exercise group had a mean age of 52±4.84 years. The control group was comparatively younger, with a mean age of 49.77±6.75 years.

Characteristics (Mean±SD)	Experimental group (n=22)	Exercise group (n=22)	Control group (n=22)
Age (years)	53±3.7	52±4.84	49.77±6.75
Height (cm)	167.60±7.52	168.04±6.17	165.63±6.82

[Table/Fig-1]: Baseline characteristics in all three groups.

The study assessed weight changes across the three groups: experimental, exercise and control. In the experimental group, the mean weight decreased from 72.82±11.88 kg at baseline to 69.93±12.47 kg at the three-month follow-up. Similarly, the exercise group exhibited a reduction in mean weight from 73.60±8.69 kg at baseline with a further decrease to 70.25±9.52 kg at three months. In contrast, the control group showed a slight increase in mean weight, rising from 79.63±10.90 kg at baseline to 80.60±10.47 kg at follow-up. Overall, the experimental and exercise groups experienced modest weight reductions, while the control group demonstrated a small weight gain over the intervention and follow-up periods [Table/Fig-2].

The results indicate the cholesterol levels across three time points (pre, post 12 weeks and post 3 months) for the control, exercise and experimental groups, each consisting of 22 participants. At baseline, the mean±SD cholesterol levels were 186.27±26.97 mg/dL in the control group, 171.23±31.04 mg/dL in the exercise group and 171.14±30.14 mg/dL in the experimental group. After three months of follow-up, the control group's cholesterol further increased to 200.18±18.24 mg/dL, while both the exercise and experimental groups showed a decline, with mean values of 162.95±24.68 mg/dL and 162.09±22.60 mg/dL, respectively [Table/Fig-3].

Time Point	Group	Mean±Std. Deviation
Pre-Total Cholesterol (TC) (mg/dL)	Control	186.27±26.97
	Exercise	171.23±31.04
	Experimental	171.14±30.14
Post 12 weeks (mg/dL)	Control	196.59±21.93
	Exercise	165.18±23.78
	Experimental	167.09±27.17
Post 3 months	Control	200.18±18.24
	Exercise	162.95±24.68
	Experimental	162.09±22.66

[Table/Fig-3]: Shows the Mean±SD of the Total Cholesterol (TC).

At baseline, the mean±SD of HDL levels were 44.91±4.97 mg/dL in the control group, 45.73±4.51 mg/dL in the exercise group and 43.55±4.39 mg/dL in the experimental group. At the three-month follow-up, the control group's HDL further declined to 40.55±4.62 mg/dL, whereas the exercise group showed a slight increase to 44.41±4.44 mg/dL and the experimental group's HDL dropped to 42.36±4.52 mg/dL [Table/Fig-4].

Time point	Group	Mean±Std. deviation
Pre HDL (mg/dL)	Control	44.91±4.97
	Exercise	45.73±4.51
	Experimental	43.55±4.39
Post 12 weeks (mg/dL)	Control	41.14±3.82
	Exercise	44.14±4.23
	Experimental	43.45±4.29
Post 3 months (mg/dL)	Control	40.55±4.62
	Exercise	44.41±4.44
	Experimental	42.36±4.52

[Table/Fig-4]: Shows the Mean±SD of the HDL.

Similarly, the results for LDL showed that at baseline, the mean±SD of LDL levels were 113.93±27.12 mg/dL in the control group, 94.96±34.83 mg/dL in the exercise group and 78.41±28.33 mg/dL in the experimental group. At the three-month follow-up, LDL levels increased in the control group to 119.29±27.41 mg/dL and in the exercise group to 92.37±33.49 mg/dL, while the experimental group experienced a slight further decrease to 76.56±25.36 mg/dL [Table/Fig-5].

The VLDL levels across the three time points (pre, post 12 weeks and post 3 months) remained relatively stable across all groups. In the control group, VLDL showed minimal fluctuations, with a slight increase at 12 weeks (37.54±9.53 mg/dL) before decreasing at three months (37.06±8.81 mg/dL). The exercise group maintained stable levels, with negligible changes from baseline (32.56±8.41) to 12 weeks (32.57±8.46) and three months (32.45±8.13). Similarly, the Experimental group showed minor variations, with VLDL levels slightly increasing at 12 weeks (33.91±10.81) before a slight rise at three months (34.18±11.00) [Table/Fig-6].

Characteristics (Mean±SD)	Experimental group (n=22)			Exercise group (n=22)			Control group (n=22)		
	Preintervention	Post 12 weeks	Post 3 months	Preintervention	Post 12 weeks	Post 3 months	Preintervention	Post 12 weeks	Post 3 months
Weight (kg)	72.82±11.88	70.28±12.39	69.93±12.47	73.60±8.69	71.47±9.24	70.25±9.52	79.63±10.90	80.17±10.83	80.60±10.47

[Table/Fig-2]: Shows the Mean±SD of weight in all three groups.

Time point	Group	Mean±Std. deviation
Pre LDL (mg/dL)	Control	113.93±27.12
	Exercise	94.96±34.83
	Experimental	78.41±28.33
Post 12 weeks (mg/dL)	Control	113.10±28.16
	Exercise	91.31±31.62
	Experimental	77.41±24.83
Post 3 months (mg/dL)	Control	119.29±27.41
	Exercise	92.37±33.49
	Experimental	76.56±25.36

[Table/Fig-5]: Shows the Mean±SD of the LDL.

Time point	Group	Mean±Std. deviation
Pre VLDL (mg/dL)	Control	37.35 ±9.08
	Exercise	37.06±8.41
	Experimental	33.77±10.89
Post 12 weeks (mg/dL)	Control	37.54±9.53
	Exercise	32.57±8.46
	Experimental	33.91±10.81
Post 3 months (mg/dL)	Control	37.06±8.81
	Exercise	32.45±8.13
	Experimental	34.18±11.00

[Table/Fig-6]: Shows the Mean±SD of the VLDL.

At baseline, the mean TG levels were 187.86±37.15 mg/dL in the control group, 176.95±38.36 mg/dL in the exercise group and 176.09±47.80 in the experimental group. At the three-month follow-up, TG levels in the control group further increased to 197.27±38.33 mg/dL, whereas the exercise group remained relatively stable at 167.91±39.45 mg/dL and the experimental group showed a further reduction to 159.55±42.25 mg/dL. These findings suggest that while TG levels increased in the control group over time, both intervention groups exhibited reductions, with the experimental group showing the most consistent decline [Table/Fig-7].

Time Point	Group	Mean±Std. Deviation
Pre Trig (mg/dL)	Control	187.86±37.15
	Exercise	176.95±38.36
	Experimental	176.09±47.80
	Total	180.30±41.09
Post 12 weeks (mg/dL)	Control	194.14±34.01
	Exercise	167.36±39.11
	Experimental	164.00±41.75
	Total	175.17±40.19
Post 3 months (mg/dL)	Control	197.27±38.33
	Exercise	167.91±39.45
	Experimental	159.55±42.25
	Total	174.91±42.66

[Table/Fig-7]: Shows the Mean±SD of the Triglycerides.

The cholesterol/HDL ratio was analysed and showed that the mean ratio at baseline was 4.182±0.85 in the control group, 3.718±0.75 in the exercise group and 3.973±0.84 in the experimental group. After 12 weeks of intervention, the ratio increased in the control group to 4.864±0.710, while remaining relatively stable in the exercise (3.705±0.756) and experimental (3.873±0.854) groups. At the three-month follow-up, the ratio in the control group further increased to 4.955±0.785, whereas the exercise group remained unchanged at 3.718±0.758 and the experimental group showed a slight increase to 3.882±0.697. These results suggest that while the cholesterol/HDL ratio increased over time in the control group, the exercise and experimental groups maintained more stable values, indicating potential benefits of the interventions [Table/Fig-8].

Time point	Group	Mean±Std. Deviation
Pre-Ratio	Control	4.182±0.853
	Exercise	3.718±0.758
	Experimental	3.973±0.844
Post 12 weeks	Control	4.864±0.710
	Exercise	3.705±0.756
	Experimental	3.873±0.854
Post 3 months	Control	4.955±0.785
	Exercise	3.718±0.758
	Experimental	3.882±0.697

[Table/Fig-8]: Shows the mean±SD of the cholesterol/HDL ratio.

The ANOVA test determines whether there are statistically significant differences in lipid profile parameters (TC, HDL, LDL, VLDL, TG and cholesterol/HDL ratio) across three time points (preintervention, post-12 weeks and post-3 months) among the three groups. The F-value represents the ratio of variance between groups to variance within groups and the p-value indicates whether the observed differences are statistically significant (p-value <0.05). The partial Eta Squared shows the effect size, indicating how much of the variance in the dependent variable is explained by the group differences. TC, HDL and TG showed significant differences over time (p<0.05), indicating that the interventions (Exercise and Experimental) had an impact on these lipid parameters. LDL showed a significant interaction effect with the group (p-value=0.003), meaning that LDL levels changed differently in each group. VLDL and cholesterol/HDL ratio also showed a significant difference (p-value=0.002), suggesting that the interventions had no substantial impact on these parameters [Table/Fig-9].

Parameter	F-value	p-value	Partial Eta Squared
Total Cholesterol (TC)	16.809	<0.001*	0.348
HDL	12.152	<0.001*	0.162
LDL	4.290	0.003*	0.120
VLDL	1.486	0.713	0.045
Triglycerides	13.389	0.001*	0.213
Cholesterol/HDL Ratio	8.765	0.002*	0.194

[Table/Fig-9]: Shows difference between groups over time by using ANOVA Analysis (*p<0.05).

Post-Hoc Tukey HSD tests were used to compare pairwise differences between the groups to determine which specific group differences are significant. The mean difference represents the change in lipid levels between two groups, while the p-value indicates whether this difference is statistically significant (p-value <0.05). TC was significantly lower in the exercise and experimental groups compared to the control group (p-value=0.001), confirming that the interventions were effective in reducing cholesterol levels. LDL was significantly lower in both the exercise and experimental groups compared to the control group (p-value=0.000), supporting the effectiveness of the interventions. TG were significantly lower in both intervention groups compared to the control group (p=0.001), reinforcing the beneficial effect of the exercise and experimental interventions. HDL showed no significant post-hoc differences, indicating that although there was a general trend of improvement, the changes were not statistically significant when compared between groups. VLDL and cholesterol/HDL ratio did not show significant pairwise differences, confirming the ANOVA findings that these parameters were not significantly affected by the interventions [Table/Fig-10].

DISCUSSION

The present study investigated the effects of EMS and RT on lipid profile parameters in individuals with T2DM. The results indicate that

Parameter	Group comparison	Mean difference	p-value (*p<0.05)	Interpretation
Total Cholesterol (TC)	Control vs. Exercise	27.894	0.001*	Exercise group significantly lower than control
	Control vs. Experimental	27.576	0.001*	Experimental group significantly lower than control
	Exercise vs. Experimental	0.318	1.000	No significant difference
HDL	Control vs. Exercise	-2.561	0.072	HDL higher in exercise but not significant
	Control vs. Experimental	-0.924	0.699	No significant difference
	Exercise vs. Experimental	1.636	0.331	No significant difference
LDL	Control vs. Exercise	35.518	<0.001*	LDL significantly lower in exercise than control
	Control vs. Experimental	42.727	<0.001*	LDL significantly lower in experimental than control
	Exercise vs. Experimental	7.209	0.477	No significant difference
VLDL	Control vs. Exercise	3.33	0.152	No significant difference
	Control vs. Experimental	3.56	0.119	No significant difference
	Exercise vs. Experimental	0.23	0.996	No significant difference
Triglycerides	Control vs. Exercise	20.5	0.001*	Triglycerides significantly lower in exercise group
	Control vs. Experimental	22.0	0.001*	Triglycerides significantly lower in experimental group
	Exercise vs. Experimental	1.5	0.989	No significant difference
Cholesterol/ HDL Ratio	Control vs. Exercise	0.60	0.093	Exercise group had a lower ratio but not significant
	Control vs. Experimental	0.53	0.128	Experimental group had a lower ratio but not significant
	Exercise vs. Experimental	0.07	0.987	No significant difference

[Table/Fig-10]: Shows Post-Hoc Analysis Results (Tukey HSD Multiple Comparisons) which compare pairwise differences between the groups to determine which specific group differences are significant.

both EMS and RT significantly improved lipid profiles in sedentary individuals with T2DM, as evidenced by the significant group × time interactions for TC, LDL-C, TG, HDL-C and the cholesterol/HDL ratio, which reject our null hypothesis. The results demonstrated a reduction in TC levels in both the EMS and RT groups, whereas the control group showed an increase over time. Present study findings are consistent with Moayedi F et al., who demonstrated that exercise training improved antioxidant defence and reduced inflammatory activity in women with diabetes dyslipidaemia [13].

RT has been documented to improve lipid metabolism by increasing enzymatic activity associated with lipid oxidation and reducing hepatic cholesterol synthesis. The observed reduction in the EMS group aligns with previous studies suggesting that neuromuscular stimulation can enhance metabolic function and lipid mobilisation in sedentary or diabetic populations [14]. However, the RT group showed a more pronounced reduction in cholesterol levels compared to the EMS group, indicating that voluntary muscle contractions may be more effective in modulating lipid metabolism.

LDL-C, commonly known as "bad cholesterol," showed a slight decrease in both the EMS and RT groups postintervention, with no significant difference between the two groups. However, the experimental group exhibited a more substantial reduction from baseline levels, with a significant difference observed within the group. This reinforces the idea that both active and passive muscle activation strategies can enhance lipid clearance and lower cardiovascular risk in individuals with diabetes. HDL-C, often referred to as "good cholesterol," showed a slight increase in the RT group but remained relatively stable in the EMS group. RT has been shown to increase HDL-C by stimulating lipoprotein lipase activity and reducing hepatic lipogenesis, while the effects of EMS on HDL-C appear to be less consistent. These findings suggest that RT may be superior in improving HDL-C levels, a key protective factor against cardiovascular disease in diabetes. Conversely, the findings of He M et al., suggested that RT can reduce LDL-C but has a negligible effect on HDL-C [15].

A significant reduction in TG levels was observed in the EMS and RT groups, with the most pronounced decrease occurring in the EMS group. Previous research indicates that EMS may enhance lipolytic enzyme activity and fat oxidation, leading to a reduction in circulating TG [25]. The observed TG-lowering effect in the RT group is also consistent with literature suggesting that RT enhances mitochondrial function and promotes fatty acid utilisation, thereby reducing TG accumulation. The control group, however, exhibited a slight increase in TG levels over time, reinforcing the importance of physical activity in lipid regulation.

The cholesterol/HDL ratio is a critical marker for cardiovascular risk, with lower values indicating a reduced risk of atherosclerosis. In this study, the RT group showed a more substantial reduction in the cholesterol/HDL ratio compared to the EMS group, suggesting that traditional RT may be more effective in modulating lipid balance. This aligns with previous findings by Messina G et al., that RT positively influences lipid profiles through increased lipoprotein turnover and improved insulin sensitivity [26]. While both EMS and RT positively influenced lipid profiles, RT had a greater overall impact on cholesterol and HDL-C levels, whereas EMS was more effective in reducing triglycerides.

The variations in lipid response between the two interventions may be due to differences in muscle activation mechanisms: RT promotes greater metabolic and hormonal adaptations, while EMS primarily enhances localised muscle contraction and circulation. These findings suggest that EMS could serve as a viable alternative or complementary approach for individuals unable to participate in conventional RT due to mobility limitations, fatigue, or co-morbid conditions. This study highlights the potential of EMS and RT as non pharmacological interventions for improving lipid profiles in individuals with T2DM. Integrating EMS into clinical practice could enhance cardiovascular risk management in diabetic populations.

Future research should explore long-term effects, optimal EMS protocols and its combination with other lifestyle interventions. Larger trials are needed to further establish EMS as a viable adjunct to traditional exercise therapies.

Limitation(s)

The convenient sampling method used in this study to recruit potential subjects may limit the generalisability of the results, while the short follow-up period restricts insights into the long-term effects of EMS and RT on lipid metabolism. Variations in diet and medication adherence were not strictly controlled, which may have influenced the outcomes. Additionally, the study did not comprehensively assess participants' overall physical activity levels or long-term adherence to the interventions, raising questions about sustainability. Individual variability in neuromuscular response to EMS may have affected the results and the absence of biochemical or molecular analysis prevents a deeper understanding of the underlying mechanisms.

CONCLUSION(S)

Both EMS and RT exert positive effects on lipid metabolism in diabetic individuals, with RT showing superior benefits in reducing cholesterol and LDL-C, while EMS was more effective in lowering TG

levels. These findings highlight the potential of EMS as an alternative training modality for individuals who may struggle with conventional RT. Further research is needed to optimise EMS protocols and explore its integration with other therapeutic approaches for improving lipid profiles and overall metabolic health in diabetes.

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REFERENCES

[1] Urrutia I, Martín-Nieto A, Martínez R, Casanovas-Marsal JO, Aguayo A, Del Olmo J, et al. Incidence of diabetes mellitus and associated risk factors in the adult population of the Basque country, Spain. Scientific Reports. 2021;11(1):3016.
[2] Bahiru E, Hsiao R, Phillipson D, Watson KE. Mechanisms and treatment of dyslipidemia in diabetes. Current Cardiology Reports. 2021;23:01-06.
[3] Yousri NA, Suhre K, Yassin E, Al-Shakaki A, Robay A, Elshafei M, et al. Metabolic and metabo-clinical signatures of type 2 diabetes, obesity, retinopathy, and dyslipidemia. Diabetes. 2022;71(2):184-205.
[4] Mehta RK, Koirala P, Mallick RL, Parajuli S, Jha R. Dyslipidemia in patients with type 2 diabetes mellitus in a tertiary care centre: A descriptive cross-sectional study. JNMA: J Nepal Med Asso. 2021;59(236):305.
[5] Einarson TR, Acs A, Ludwig C, Panton UH. Prevalence of cardiovascular disease in type 2 diabetes: A systematic literature review of scientific evidence from across the world in 2007–2017. Cardiovascular Diabetology. 2018;17:01-09.
[6] Ginsberg HN, Zhang YL, Hernandez-Ono A. Metabolic syndrome: Focus on dyslipidemia. Obesity. 2006;14(2S):41S.
[7] Tomlinson B, Patil NG, Fok M, Lam CW. Managing dyslipidemia in patients with Type 2 diabetes. Expert Opinion on Pharmacotherapy. 2021;22(16):2221-34.
[8] Banach M, Surma S, Reiner Z, Katsiki N, Penson PE, Fras Z, et al. Personalized management of dyslipidemias in patients with diabetes—it is time for a new approach (2022). Cardiovascular Diabetology. 2022;21(1):263.
[9] Feingold KR, Grunfeld C. Diabetes and dyslipidemia. In Diabetes and Cardiovascular Disease 2023 Jun 2 (pp. 425-472). Cham: Springer International Publishing.
[10] Karter AJ, Parker MM, Solomon MD, Lyles CR, Adams AS, Moffet HH, et al. Effect of out-of-pocket cost on medication initiation, adherence, and persistence among patients with type 2 diabetes: The Diabetes Study of Northern California (DISTANCE). Health Ser Res. 2018;53(2):1227-47.
[11] Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: Lipid modification to reduce cardiovascular risk: The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS). European Heart J. 2020;41(1):111-88.
[12] Grundy SM, Stone NJ, Bailey AL, Beam C, Birtcher KK, Blumenthal RS, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. J Am Coll Cardiol. 2019;73(24):e285-e350.
[13] Moayedi F, Taghian F, Dehkordi KJ, Hosseini SA. Cumulative effects of exercise training and consumption of propolis on managing diabetic dyslipidemia in adult women: A single-blind, randomized, controlled trial with pre–post-intervention assessments. J Physiol Sci. 2023;73(1):17.
[14] Ye S, Stetter S, McCully KK. Muscle metabolism during multiple muscle stimulation using an affordable equipment. J Funct Morphol Kinesiol. 2024;9(4):248.
[15] He M, Hu S, Wang J, Wang J, Găman MA, Hariri Z, et al. Effect of resistance training on lipid profile in postmenopausal women: A systematic review and meta-analysis of randomized controlled trials. Eur J Obstet Gynecol Reprod Biol. 2023;288:18-28.
[16] Sharma D, Shenoy S, Singh J. Effect of electrical stimulation on blood glucose level and lipid profile of sedentary type 2 diabetic patients. Int J Diabe Develop Countr. 2010;30(4):194.
[17] Galvan MJ, Sanchez MJ, McAinch AJ, Covington JD, Boyle JB, Bajpeyi S. Four weeks of electrical stimulation improves glucose tolerance in a sedentary overweight or obese Hispanic population. Endocr Connect. 2022;11(2):e210533.
[18] Sanchez MJ, Mossayebi A, Sigaroodi S, Apaflo JN, Galvan MJ, Min K, et al. Effects of neuromuscular electrical stimulation on glycemic control: A systematic review and meta-analysis. Front Endocrinol. 2023;14:1222532.
[19] Johns Hopkins Medicine. Lipid Panel [Internet]. Baltimore (MD): Johns Hopkins Medicine; 2021 [Internet] [cited 2025 Mar 3]. Available from: https://www.hopkinsmedicine.org/health/treatment-tests-and-therapies/lipid-panel.
[20] Booth M. Assessment of physical activity: An international perspective. Research Quarterly for Exercise and Sport. 2000;71(sup2):114-20.
[21] Gogtay NJ. Principles of sample size calculation. Indian J Ophthalmol. 2010;58(6):517-18.
[22] Crowe L, Caulfield B. Aerobic neuromuscular electrical stimulation—an emerging technology to improve hemoglobin A1c in type 2 diabetes mellitus: Results of a pilot study. BMJ Open. 2012;2012:e000219.
[23] Brzycki M. Strength testing—predicting a one-rep max from reps-to-fatigue. Journal of Physical Education, Recreation & Dance. 1993;64(1):88-90.
[24] Misra A, Alappan NK, Vikram NK, Goel K, Gupta N, Mittal K, et al. Effect of supervised progressive resistance-exercise training protocol on insulin sensitivity, glycaemia, lipids, and body composition in Asian Indians with type 2 diabetes. Diabetes Care. 2008;31(7):1282-87.
[25] Miyamoto T, Fukuda K, Kimura T, Matsubara Y, Tsuda K, Moritani T. Effect of percutaneous electrical muscle stimulation on postprandial hyperglycaemia in type 2 diabetes. Diabetes Research and Clinical Practice. 2012;96(3):306-12.
[26] Messina G, Alioto A, Parisi MC, Mingrino O, Di Corrado D, Crescimanno C, et al. Experimental study on physical exercise in diabetes: Pathophysiology and therapeutic effects. Eur J Transl Myol. 2023;33(4):11560.

PARTICULARS OF CONTRIBUTORS:
1. Associate Professor, Department of Physiotherapy, G D Goenka University, Gurugram, Haryana, India.
2. Associate Director, Department of Internal Medicine, Max Super Specialty Hospital, Gurugram, Haryana, India.
3. Associate Professor, Department of Physiotherapy, G D Goenka University, Gurugram, Haryana, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Kamran Ali,
Associate Professor, Department of Physiotherapy, G D Goenka University,
Gurugram-122103, Haryana, India.
E-mail: k.alisportsphysio@gmail.com

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