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Effect of high intensity interval training on arterial stiffness in obese hypertensive women: a randomized controlled trial

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Abstract. – OBJECTIVE: High-intensity interval training (HIIT) has been linked to a lower risk of cardiovascular disease and mortality. The study's overarching goal is to evaluate the impact of HIIT on arterial stiffness in obese hypertensive women.

PATIENTS AND METHODS: Sixty obese hypertensive women aged between 40-50 years were randomized to group A (Intervention group, n = 30) or group B (Control group, n = 30). Intervention group received HIIT (4 minutes of cycling at 85-90% of peak HR interspersed with 3-minute active recovery time at 60 - 70% of peak HR, three times per week). Arteriovenous stiffness indicators, the augmentation index corrected for heart rate 75 (AIx@75HR), and oscillometric pulse wave velocity (o-PWV), as well as cardio-metabolic parameters, were assessed before and after 12 weeks of treatment.

RESULTS: Finding between-group analysis showed a significant difference in AIx@75HR (95% CI: -8.45 to 0.30) , o-PWV (95% CI: -1.14 to 0.15), total cholesterol, (95% CI: -31.25 to -1.12), HDL-cholesterol (95% CI: 8.92 to 0.94), LDL-cholesterol (95% CI: -25.35 to -0.06) , and triglycerides (95% CI: -53.58 to -2.51).

CONCLUSIONS: High-intensity interval training for 12 weeks has a favorable effect on arterial stiffness in obese hypertensive women and lowers associated cardio-metabolic risk factors.

Key Words:
Arterial stiffness, High-intensity interval training, Hypertension, Obesity, Women.

Introduction

Hypertension is considered one of the risk factors for cardiovascular disease and the prominent cause of mortality worldwide[1]. Despite significant progress in the management of hypertension, it continues to be a major medical issue with an increasing global prevalence[2]. Hypertension is a multifactorial condition in which arterial stiffness is one of its manifestations. High central (aortic) arterial stiffness has been recognized as a risk factor for cardiovascular events and death worldwide[4].

Vascular aging, obesity, hypertension, and diabetes lead to rigid arteries and endothelial dysfunction, which may contribute to the development of cardiovascular disease[5]. It has been reported that the release of metalloproteinases, the fragmentation of elastin sheets, and the activation of inflammatory reactions involving calcium and collagen deposition induced decline in the arterial elastic component which is a potent mechanism underlying the mechanical damage to the arterial walls[6,7]. In addition, patients with hypercholesterolemia have more rigid blood vessels than comparable controls[8]. Extrinsic risk factors including the lifestyle behavior such as physical inactivity contribute to vascular function impairment, atherosclerosis, and cardiovascular diseases[9]. Therefore, effective therapeutic approaches to improve arterial function are critical to combating hypertension complications and reducing the risk of cardiovascular disease in the hypertensive population[10].

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Currently, estimation of pulse wave velocity (PWV) is the most effective method for determining arterial stiffness[11]. PWV is widely recognized as an independent predictive marker for cardiovascular disease development[12]. Recently, it has become possible to assess PWV in clinical practice using non-invasive, operator-independent oscillometric methods[13]. Specific devices, such as the widely used Mobil-O-Graph, have been developed to estimate PWV by combining cuff oscillometry and pulse wave analysis on a single oscillometric blood pressure measurement[14].

Exercise is nonpharmaceutical intervention is known to induce ample of cardiovascular benefit due to pressure loading effect, which increases nitric oxide generation by endothelial cells, improves vasodilation, and decreases vascular resistance[15,16]. The most significant barrier for sedentary people to engaging in long-term aerobic training is the lack of time. In recent years, high-intensity interval training (HIIT) has steadily gained popularity. HIIT includes alternating short periods of high-intensity activity with intervals of recovery time or gentle exercise[17]. Pescatello et al[18] suggest that vigorous-intensity aerobic exercise training should be added to future exercise prescription guidelines for people with high blood pressure. HIIT has been demonstrated to reduce arterial stiffness in normotensives[19,20], men with stage 1 hypertension[21] and sedentary hypertensives[22]. However, the effectiveness of HIIT on arterial stiffness in hypertensive individuals is less clear and showed contradictory findings due to methodological differences. The lack of data on the favorable effects of HIIT on arterial stiffness in the hypertensive patients with high body composition emphasizes the need for more research to better understand mechanistic effect and enhance therapeutic outcomes for such individuals. Furthermore, the efficacy, feasibility, and safety of HIIT should be established in those with high risk factors for cardiovascular disease, such as inactive, obese, and hypertensive patients. Therefore, the aim of this study was to evaluate if the mechanistic effect of HIIT would affect arterial stiffness parameters in sedentary obese hypertensive women. It was hypothesized that HIIT would reduce arterial stiffness.

Patients and Methods

Study Design and Participants

In this randomized controlled trial, sixty hypertensive women between the ages of 40 and 50 were examined and randomly assigned to one of two groups: 1) 12-week of high-intensity interval training or 2) a control group. A computer-based random number generator determines the group assignment. Patients were chosen to participate in this study if they met the eligibility requirements, which included having stable primary hypertension that was pharmacologically controlled (systolic blood pressure 140 mmHg and diastolic blood pressure 90 mmHg), without complications for at least three months, and without modification during the intervention or follow-up. In addition, patients must have a body mass index (BMI) between 0-39.9 kg/m^2, report being sedentary, and have not engaged in any weight loss activity in the previous six months. Participants were excluded if they had unstable cardiac conditions, uncontrolled hypertension, diabetes, pulmonary disease, renal dysfunction, or musculoskeletal problems that made it difficult to exercise.

All procedures were carried out in accordance with the Declaration of Helsinki and protocols approved by Cairo University's Faculty of Physical Therapy's Institutional Research Board (P.T.REC/012/003094). The study was conducted at Cairo University's Kasr El Aini teaching hospital's outpatient clinic. The trial has been registered on Clinicaltrials.gov with registration number NCT04862754.

Training Protocol

Heart rate (HR) and rate of perceived exertion (RPE) were measured and recorded throughout each exercise session using a heart rate monitor (Polar, FT1, Kempele, Finland) and the Borg 6-20 scale, respectively. Each HIIT training session began with a 10-minute warm-up at 50% of peak HR, followed by four bouts of four minutes (4×4) of cycling at 85-90% of peak HR and an RPE of 15-17 on the Borg scale using an electronic bicycle ergometer (Biodex LBC, Biodex Inc., New York, NY, USA). Training was interspersed with 3-minute active recovery time at 60-70% of peak HR and RPE of 11-13 on the Borg scale. The exercise intensity in the first two weeks was 85% of peak HR, and the recovery period was 60% of peak HR. After two weeks, the exercise intensity increased to 90% of peak HR while the recovery intensity increased to 70% of peak HR (active recovery). The session ended with a 10-minute cool-down period. For a period of 12 weeks, the exercise protocol was conducted three times each week. A submaximal incremental exercise stress test was used in the cardiopulmonary unit to

determine each subject's peak HR. Each subject underwent symptom-limited cardiopulmonary exercise testing using an electronically upright cycle ergometer (Excalibur Sport V2.0; Lode BV, Groningen, The Netherlands). The ramp protocol was carried out until the volitional exhaustion. For proper fitting, the subjects were required to first sit for 3 minutes while cycling at 0 watts (W), and then they had to warm up for 3 minutes while cycling at 40 W. After that, the workload was raised by 15-20 W every minute until volitional fatigue was reached. The protocol ends with a 3-min active recovery at 40 W and a 3-min passive recovery at 0 W[24]. Throughout the test, each subject was told to keep their speed at 60 revolutions per minute (RPM). Peak heart rates were measured when subjects met at least two of the following criteria: (a) being unable to pedal at a rate of 60 revolutions per minute for longer than 5 seconds with verbal encouragement; (b) their perceived exertion score reached ≥ 19 on the Borg scale; or (c) volitional fatigue. Throughout the entire test, HR and RPE were monitored simultaneously. The peak HR was reviewed monthly, and the workload was adjusted to ensure a consistent training stimulus. The exercise intensity was adjusted according to the American College of Sports Medicine's recommendations[18]. The control group did not participate in any supervised exercise and instead followed their pharmaceutical and nutritional recommendations.

Outcome Measurement

Changes in arterial stiffness indices were the primary outcomes, while changes in cardiometabolic parameters were secondary outcomes. Measurements were taken at the same time each morning at baseline and 48 hours after the last exercise session. The arterial stiffness indices were measured by a single experienced operator, and blood analysis was performed by a single experienced analyst. Outcome assessors were blind to group allocation.

Arterial Stiffness Indices

The oscillometric pulse wave velocity (o-PWV), augmentation index (AIx@75HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were all assessed utilizing a non-invasive blood pressure measurement device (Mobil-O-Graph, I.E.M., GmbH Stolberg, Germany). It is a reliable tool that measured the pulse wave analysis and arterial stiffness through estimated measure of both brachial and central blood pressures as well as pulse wave velocity in a single measuring cycle[26]. This device is a cuff-based, non-invasive oscillometric device that uses a transfer function from brachial pressure waves and has been validated in accordance with European Society of Hypertension standards[25].

Three hours prior to the assessments, the participants were encouraged to refrain from eating or intake caffeine-beverages. Participants were instructed to relax for at least 5 minutes at room temperature in a darkened room with no light and no loud noise. Participants were instructed to sit comfortably with their backs supported, their legs uncrossed, and their upper arm were placed at heart level while being measured. The non-dominant arm was used for measurements. Measurement was conducted in line with Taskforce III recommendations on clinical artery stiffness applications[27].

When the data is read out, each measurement is checked for errors. If the data quality is rated 3 or 4 by a software-based automatic quality check (HMS Client-Server v4.7.3, I.E.M. GmbH, Aachen, Germany), the value is removed. If this was the case, another measurement was taken, for a total of six records.

Blood Analysis (Metabolic Parameters)

The patient is advised to fast at least eight hours before the testing. A 6-mL blood sample was drawn at the same time of day and tested for plasma triglycerides (TG), total cholesterol (TC), high-density lipoprotein (HDL), and low-density lipoprotein (LDL) using a biochemical analyzer before and after the intervention[28]. Blood samples were taken 24 hours before the start of the exercise training and 48 hours after the last exercise session.

Statistical Analysis

The sample size was calculated based on the reduction in pulse wave velocity, which was the primary outcome of this study. Using G*power 3.1 software (Franz Faul, Universitat Kiel, Germany) in which the power set at 95 percent, a p-value of 0.05, and an effect size of 0.76[29], a sample size of 21 patients in each group was required. The sample was raised to 30 in each group to account for the dropout rate. The study's consort diagram is described in Figure 1.

Statistical analyses were performed using SPSS version 25.0 (OBM Corp., Armonk, NY, USA). The Shapiro-Wilk and Levene's tests were used to check normality and homogeneity of variance in which no violations were found for any of the dependent variables. A two-way multivariate

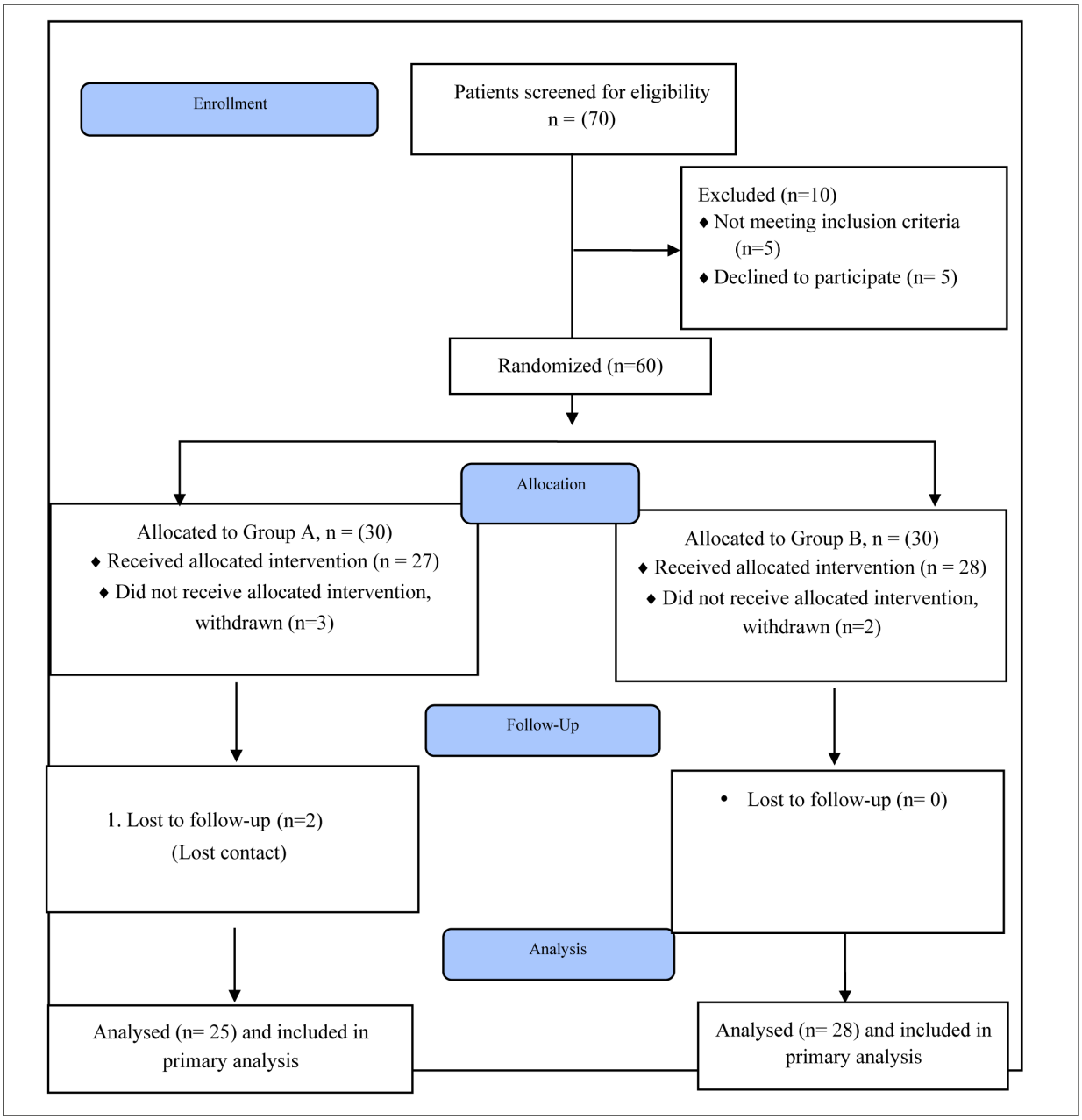

Figure 1. Consort diagram for the study.

mixed model analysis of variance (MANOVA) was used to estimate the main effect between and within the group. When the MANOVA revealed a significant time-group interaction effect, follow-up univariate ANOVAs (two-way mixed model) was performed. Pearson's correlation coefficients were used to calculate the correlations between the variables. Multiple linear regression analyses were carried to identify the independent predictors of arterial stiffness indices. In these analyses, the independent variables were chosen based on their associations with the dependent variable. The statistical significance level was set at p-value < 0.05.

Results

Table I displays the demographic and clinical characteristics of patients. The analysis of the data

Table I. Baseline demographic and clinical characteristics of patients.

Characteristics	HIIT Group Mean ± SD	Control Group Mean ± SD	Mean difference	95% CI	*p*-value
Age (years)	48.32 ± 4.48	48.92 ± 3.60	-0.59	(-2.86, 1.66)	0.59
Height (cm)	161.89 ± 7.00	162.64 ± 6.22	-0.74	(-4.42, 2.92)	0.68
Weight (kg)	88.96 ± 5.91	92.18 ± 5.77	-3.21	(-6.44, 0.01)	0.05
BMI (kg/m^2)	33.90 ± 2.58	34.90 ± 1.53	-0.99	(-2.19, 0.19)	0.09
Total cholesterol (mg/dL)	203.89 ± 30.93	205.84 ± 38.40	1.94	(-17.19, 21.08)	0.83 [a]
HDL-cholesterol (mg/dL)	37.10 ± 4.80	38.12 ± 6.88	1.01	(-2.23, 4.25)	0.53 [a]
LDL-cholesterol (mg/dL)	131.96 ± 22.73	139.44 ± 18.93	7.47	(-4.14, 19.09)	0.20 [a]
Triglycerides (mg/dL)	177.97 ± 48.83	179.95 ± 44.07	1.98	(-23.79, 27.75)	0.87 [a]
SBP (mmHg)	147.89 ± 6.08	149.12 ± 5.62	1.22	(-2.01, 4.47)	0.45 [a]
DBP (mmHg)	92.39 ± 5.78	93.16 ± 5.51	0.76	(-2.36, 3.89)	0.62 [a]
AIx@75 (%)	34.76 ± 6.60	36.11 ± 6.32	1.35	(-2.22, 4.93)	0.45 [a]
o-PWV (m/s)	8.45 ± 0.99	8.37 ± 0.91	-0.08	(-0.61, 0.44)	0.74 [a]
Medications					
ACEI/ARB (%)	46.7	60			0.13 *
β-Blocker (%)	40	33.3			
Calcium channel blocker (%)	13.3	6.7			

BMI, body mass index; HLD, high-density lipoprotein; LDL, low-density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure; AIx@75HR, augmentation index; o-PWV, oscillometric pulse wave velocity; ACEI, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers; SD, standard deviation; CI, confidence interval; [a], adjustment for pairwise multiple comparison: Bonferroni; *, the value is calculated using the Kruskal-Wallis test, level of significance at $p < 0.05$.

revealed no significant differences between the two groups at baseline ($p > 0.05$).

Repeated measures MANOVA revealed a substantial main effect of time (Wilks' $\Lambda = 0.09$, $F(8, 44) = 53.34$, $p \leq 0.001$, $\eta^2 = 0.91$), treatment (Wilks' $\Lambda = 0.35$, $F(8, 44) = 10.22$, $p \leq 0.001$, $\eta^2 = 0.65$), and a significant time-treatment interaction (Wilks' $\Lambda = 0.12$, $F(8, 44) = 38.47$, $p \leq 0.001$, $\eta^2 = 0.87$). Following-up univariate ANOVAs revealed a significant change in Total cholesterol (TC), $F(1, 51) = 108.38$, $p < 0.001$, $\eta^2 = 0.68$; HDL-cholesterol, $F(1, 51) = 135.46$, $p < 0.001$, $\eta^2 = 0.72$; LDL-cholesterol, $F(1, 51) = 51.91$, $p < 0.001$, $\eta^2 = 0.50$; triglycerides, $F(1, 51) = 75.30$, $p < 0.001$, $\eta^2 = 0.59$; SBP, $F(1, 51) = 158.27$, $p < 0.001$, $\eta^2 = 0.75$; DBP, $F(1, 51) = 118.57$, $p < 0.001$, $\eta^2 = 0.69$; AIx@75HR, $F(1, 51) = 44.25$, $p < 0.001$, $\eta^2 = 0.46$; and o-PWV, $F(1, 51) = 54.82$, $p < 0.001$, $\eta^2 = 0.52$.

The HIIT group experienced significant reductions in TC, LDL-cholesterol, triglycerides, SBP, DBP, AIx@75HR, and o-PWV after the intervention, as well as an increment in HDL-cholesterol ($p < 0.001$). Multiple comparison analyses demonstrated a substantial difference within and between the HITT and control groups in arterial stiffness indices and cardiometabolic parameters , where the mean differences at 95% confidence interval were (-31.25, -1.12) for TC; (8.92, 0.94) for HDL-cholesterol; (-25.35, -0.06) for LDL-cholesterol; (-53.58, -2.51) for Triglycerides; (-17.96, -2.64) for SBP; (-8.00, -1.32) for DBP; (-8.45, 0.30) for AIx@75HR; and (-1.14, 0.15) for o-PWV, respectively, as shown in Tables II and III.

Table IV demonstrates the correlation between arterial stiffness indices and cardio-metabolic features. o-PWV was significantly correlated with TC, HDL-cholesterol, LDL-cholesterol triglycerides, SBP, and AIx@75HR. Also, AIx@75HR was significantly correlated with TC, HDL-cholesterol, LDL-cholesterol , triglycerides, and o-PWV. We performed multiple linear regression analyses to determine the independent predictors of arterial stiffness indices (Table V). In the first model, we included TC, HDL-cholesterol, LDL-cholesterol, triglycerides, SBP, AIx@75HR at baseline, AIx@75HR, and o-PWV at baseline as independent variables. In the best-fit model, LDL-cholesterol (β-coefficient = 0.21, $p = 0.012$), SBP (β-coefficient = 0.39, $p \leq 0.001$), and o-PWV at baseline (β-coefficient = 0.64, $p \leq 0.001$), were all significantly associated with o-PWV. Whereas, in the second model, we included TC, HDL-cholesterol, LDL-cholesterol, triglycerides, SBP, DBP, o-PWV at baseline, o-PWV, and AIx@75HR at baseline as independent variables. LDL-cholesterol (β-coefficient = 0.23, $p = 0.017$), SBP (β-coefficient = 0.40, $p \leq 0.001$), and AIx@75HR at baseline (β-coefficient = 0.53, $p \leq 0.001$), were all significantly associated with AIx@75HR.

Table II. Arterial stiffness indices and cardio-metabolic features post-intervention [a].

Characteristics	HIIT Group Mean ± SD	Control Group Mean ± SD	Mean difference	95% CI	*p*-value
Total cholesterol (mg/dL)	172.64 ± 26.82	204.72 ± 35.88	32.07	(14.72, 49.43)	< 0.001
HDL-cholesterol (mg/dL)	46.03 ± 4.48	39.06 ± 5.28	-6.96	(-9.65, -4.26)	< 0.001
LDL-cholesterol (mg/dL)	106.61 ± 16.58	139.37 ± 14.78	32.76	(24.05, 41.47)	< 0.001
Triglycerides (mg/dL)	124.39 ± 41.47	177.44 ± 43.55	53.04	(29.59, 76.50)	< 0.001
SBP (mmHg)	129.92 ± 5.71	146.48 ± 5.02	16.55	(13.56, 19.53)	< 0.001
DBP (mmHg)	84.39 ± 3.84	91.84 ± 3.64	7.44	(5.37, 9.52)	< 0.001
AIx@75 (%)	26.31 ± 6.08	36.41 ± 5.91	10.10	(6.78, 13.42)	< 0.001
o-PWV (m/s)	7.31 ± 1.03	8.52 ± 0.84	1.20	(0.68, 1.73)	< 0.001

HLD, high-density lipoprotein; LDL, low-density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure; AIx@75, Augmentation index corrected for 75 beats per minute; o-PWV, oscillometric pulse wave velocity; SD, standard deviation; CI, confidence interval; [a], adjustment for pairwise multiple comparison: Bonferroni; level of significance at $p < 0.05$.

Table III. Arterial stiffness indices and cardio-metabolic features pre-and post-intervention [a].

Characteristics		Pre-intervention Mean ± SD	Post-intervention Mean ± SD	Mean difference	95% CI	*p*-value
Total cholesterol (mg/dL)	HIIT Group	203.89 ± 30.93	172.64 ± 26.82	-31.25	(-35.53, -6.96)	< 0.001
	Control Group	205.84 ± 38.40	204.72 ± 35.88	-1.12	(-5.65, 3.41)	0.62
HDL-cholesterol (mg/dL)	HIIT Group	37.10 ± 4.80	46.03 ± 4.48	8.92	(7.75, 10.09)	< 0.001
	Control Group	38.12 ± 6.88	39.06 ± 5.28	0.94	(-0.29, 2.18)	0.13
LDL-cholesterol (mg/dL)	HIIT Group	131.96 ± 22.73	106.61 ± 16.58	-25.35	(-30.21, -0.48)	< 0.001
	Control Group	139.44 ± 18.93	139.37 ± 14.78	-0.06	(-5.20, 5.08)	0.98
Triglycerides (mg/dL)	HIIT Group	177.97 ± 48.83	124.39 ± 41.47	-53.58	(-62.49, -4.67)	< 0.001
	Control Group	179.95 ± 44.07	177.44 ± 43.55	-2.51	(-11.95, 6.91)	0.59
SBP (mmHg)	HIIT Group	147.89 ± 6.08	129.92 ± 5.71	-17.96	(-20.22, -5.70)	< 0.001
	Control Group	149.12 ± 5.62	146.48 ± 5.02	-2.64	(-5.03, -0.25)	0.03
DBP (mmHg)	HIIT Group	92.39 ± 5.78	84.39 ± 3.84	-8.00	(-9.18, -6.82)	< 0.001
	Control Group	93.16 ± 5.51	91.84 ± 3.64	-1.32	(-2.56, -.07)	0.03
AIx@75 (%)	HIIT Group	34.76 ± 6.60	26.31 ± 6.08	-8.45	(-10.14, -6.76)	< 0.001
	Control Group	36.11 ± 6.32	36.41 ± 5.91	0.30	(-1.48, 2.08)	0.73
o-PWV (m/s)	HIIT Group	8.45 ± 0.99	7.31 ± 1.03	-1.14	(-1.32, -0.95)	< 0.001
	Control Group	8.37 ± 0.91	8.52 ± 0.84	0.15	(-0.04, 0.34)	0.12

HLD, high-density lipoprotein; LDL, low-density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure; AIx@75, Augmentation index corrected for 75 beats per minute; o-PWV, oscillometric pulse wave velocity; SD, standard deviation; CI, confidence interval; [a], adjustment for pairwise multiple comparison: Bonferroni; level of significance at $p < 0.05$.

Table IV. Correlations between arterial stiffness indices and Cardio-metabolic features.

Variables	PWA (m/s)		AIx@75 (%)	
	r	*p*-value	*r*	*p*-value
Total cholesterol (mg/dL)	0.32	0.02	0.42	0.002
HDL-cholesterol (mg/dL)	-0.38	0.006	-0.47	< 0.001
LDL-cholesterol (mg/dL)	0.45	0.001	0.53	< 0.001
Triglycerides (mg/dL)	0.33	0.016	0.37	0.005
SBP (mmHg)	0.61	< 0.001	0.64	< 0.001
DBP (mmHg)	**	**	0.37	0.006
AIx@75 at baseline (%)	0.34	0.012	0.63	< 0.001
AIx@75 (%)	0.64	< 0.001	1.00	1.00
o-PWV at baseline (m/s)	0.70	< 0.001	0.31	0.02
o-PWV (m/s)	1.00	1.00	0.64	< 0.001

HLD, high-density lipoprotein; LDL, low-density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure; AIx@75, Augmentation index corrected for 75 beats per minute; o-PWV, oscillometric pulse wave velocity; *r*, correlation coefficient; **, non-significant; level of significance at $p < 0.05$.

Table V. Predictors of change in arterial stiffness indices by multiple regression analysis.

Variables in the model	β-coefficients	95% CI	*p*-value
Model 1: change in o-PWV ($R^2 = 0.793$, $p < 0.001$)			
o-PWV at baseline (m/s)	0.649	(0.612, 0.926)	< 0.001
SBP (mmHg)	0.393	(0.026, 0.063)	< 0.001
LDL-cholesterol (mg/dL)	0.213	(0.002, 0.019)	0.012
Model 2: change in AIx ($R^2 = 0.705$, $p < 0.001$)			
AIx@75 at baseline (%)	0.532	(0.459, 0.833)	< 0.001
SBP (mmHg)	0.407	(0.170, 0.472)	< 0.001
LDL-cholesterol (mg/dL)	0.231	(0.015, 0.145)	0.017

HLD, high-density lipoprotein; LDL, low-density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure; AIx@75, Augmentation index corrected for 75 beats per minute; o-PWV, oscillometric pulse wave velocity; CI, confidence interval; level of significance at $p < 0.05$.

Discussion

The main finding of this study highlights the beneficial effect of HITT on lowering arterial stiffness in obese hypertensive women. Furthermore, HIIT resulted in significant improvements in SBP, DPB, TC, HDL-cholesterol, LDL-cholesterol, and triglycerides.

According to epidemiological data[30], systolic blood pressure rises by 14% from early to late adulthood (i.e., 20-90 years), while AIx@75HR rises by five times and PWV rises by two times. PWV has been shown to be an independent predictor of systolic blood pressure increase over time[31]. A 1-m/s rise in brachial-ankle pulse wave velocity is related to a 12% rise in the incidence of cardiovascular events in subjects with hypertension[32]. Therefore, lowering both arterial stiffness and blood pressure in high-risk populations may reduce the risk of cardiovascular disease. Arterial pulse wave contour assessments are thought to be more sensitive than conventional brachial BP measurements in detecting changes in vascular structure and function associated with aging or pathological conditions like hypertension[33].

Aerobic exercise has been shown to reduce sympathetic nervous system overactivity, increase baroreflex sensitivity, decrease mRNA and protein expression of angiotensin II type 1 receptor, increase resting arterial diameter, and improve local vascular function, all of which have a positive impact on peripheral resistance[34-36].

Several mechanisms have been proposed to explain why exercise lowers o-PWV, AIx@75HR, and blood pressure. One possible mechanism for these changes is an increase in endothelial nitric oxide synthase activity, which leads to increased nitric oxide bioavailability, a potent vasodilator involved in lowering arterial stiffness[37]. Factors that contribute to arterial stiffness include endothelial dysfunction, elastic matrix degradation, smooth muscle cell hypertrophy and hyperplasia, and elevated collagen content[38]. It is possible that the training-induced improvements in blood pressure and o-PWV are attributable to better endothelial function, as evidenced by increased blood levels of nitrite/nitrate and reduced endothelin[39]. In addition, endothelial-dependent dilation may be augmented by improved endothelial function, which lowers vascular tone and resistance in peripheral arteries, thereby lowering both systolic and diastolic blood pressure[40].

Because of the high exercise intensity, HIIT has been observed to raise plasma nitrite/nitrate and endothelin-1 levels during rest, activity, and recovery, which may be attributed to increased shear stress[41]. The amount of wall distension caused by a given shear stress influences the change in arterial stiffness caused by mechano-biochemical signaling, resulting in vasodilation[42]. When hypertensive individuals exercise at a high intensity for an extended period, the resistance to blood flow in their peripheral arteries is reduced even further.

Our results were consistent with those of Bahmanbeglou et al[21], who examined the effects of two HIIT protocols on males with Stage 1 hypertension. The first protocol was long-duration HIIT (4 minutes of activity at 75% to 90% of VO_2 peak followed by 4 minutes of active recovery for four repetitions), while the second protocol was short-duration HIIT (30 seconds of activity at 80% to 100% of VO_2 peak followed by 27 repetitions). Authors found that regardless of HIIT intensity or duration, patients' systolic blood pressure, triglyceride levels, and inflammatory markers decreased. On the other hand, PWV was significantly increased after short-duration HIIT and was linked

to exercise intensity. The authors' assumption is supported by the fact that rapid increases in shear stress rate occurred during short-duration HIIT 27 times more frequently than during long-duration HIIT.

Guimarães et al[22] reported that both continuous and interval training exercises were helpful for blood pressure control after 16 weeks of training with two sessions per week, but only interval training significantly reduced PWV and AIx@75HR in treated hypertensive participants. In addition, several studies[19,43,44] have shown that HIIT improves AIx@75HR in adults with abdominal obesity[43], PWV in young and normotensive women with a high familial risk of hypertension, as well as young male participants[19,44]. The findings of the study are consistent with previous study[33,45] of patients with metabolic syndrome, who underwent six months of intense aerobic exercise , which result in reduction of arterial stiffness, lower systolic and diastolic pressures and a 7% reduction in the AIx@75HR[33]. Furthermore, 12 weeks of Taekwondo training reduced arterial stiffness and elevated blood catecholamine levels in postmenopausal women with stage-2 hypertension[45].

On the other hand, Ramos et al[46] compared the effects of two different volumes of HIIT and moderate-intensity continuous training on arterial stiffness in patients with metabolic syndrome. They found that both training protocols improved SBP but had no effect on arterial stiffness indices (Carotid-femoral pulse wave velocity and AIx@75HR) in all training regimens. These inconsistent findings could be attributed to the fact that the majority of the training in their study was unsupervised, and not all patients completed the entire schedule of training sessions.

Regarding the influence of HIIT on cardio-metabolic parameters, several studies[19,41] have demonstrated that HIIT is a time-efficient method for improving health-related indicators such as lipid profiles in various populations. HIIT regimens with a weekly commitment have been shown to improve blood lipids in patients who had an abnormal blood lipid profile[47,48]. Alvarez et al[49] found that HIIT was effective in normalizing the TC, LDL, and HDL of those with hyperlipidemia and those with hyperlipidemia combined with hyperglycemia to values that were equivalent to the healthy control group. Exercise is thought to lower very low-density lipoprotein and TG levels while also reducing the availability of cholesteryl ester exchanges between HDL and LDL, resulting in higher HDL levels and smaller LDL particles[50]. In terms of the regression analysis, o-PWV correlated significantly with LDL-cholesterol, SBP, and AIx@75HR, whereas AIx@75HR correlated significantly with HDL-cholesterol, LDL-cholesterol, SBP, and o-PWV. This is consistent with the findings of Wang et al[51], who found that TC, TG, and LDL cholesterol levels were all positively correlated to o-PWV, while HDL cholesterol levels were inversely related.

Limitations

This study has some limitations, including the use of a convenience sampling method that did not adequately represent the entire population. Furthermore, future research study is required to assess the long-term effects of HIIT.

Conclusions

High-intensity interval training for 12 weeks reduces cardiometabolic risk factors and improves arterial stiffness indices in obese hypertensive women. This study supports the inclusion of high-intensity interval training in the treatment of obese hypertensive women in order to reduce their risk of cardiovascular disease.

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Conflict of Interest

There are no conflicts of interest declared by the authors.

Informed Consent

All subjects who participated in the study gave their informed consent.

Ethics Approval

The Institutional Research Board of Cairo University's Faculty of Physical Therapy (P.T.REC/012/003094) approved the study methodology, and it was carried out in compliance with the Declaration of Helsinki's principles.

Authors' Contributions

All authors contributed to the design, patient selection and implementation of treatments, acquisition and reviewing of data, statistical analysis, interpretation, writing, and revision of the manuscript.

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Data Availability

The corresponding author can provide the study's datasets upon request.

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